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Effectiveness of non-invasive positive pressure ventilation differs between decompensated chronic restrictive and obstructive pulmonary disease patients

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Abstract *Objective:* To compare the efficiency of non-invasive positive pressure ventilation (NPPV) in decompensated patients with either chronic obstructive pulmonary disease (COPD) or chronic restrictive pulmonary disease. *Design:* Retrospective study. *Setting:* A 17-bed intensive care unit in a university teaching hospital. *Setting:* Sixty-four patients with COPD (age: 70±13 years, sex ratio: 37 male to 27 female patients, forced expiratory volume in 1 s: 31±13% predicted) and 20 patients with chronic restrictive pulmonary disease (age: 75±9 years, sex ratio: 9 male to 11 female patients, total lung capacity: 57±17% predicted) consecutively treated with NPPV (facial mask, pressure support ventilation (PSV) ± PEEP) for acute respiratory failure. *Measurements and results:* There were no statistically significant differences between COPD and patients with chronic restrictive pulmonary disease in terms of cause of exacerbation, use of oxygen therapy or NPPV at home, severity of acute respiratory failure (ARF), mean delay from intensive care admission to initiation of NPPV and total duration of NPPV. Patients with chronic restrictive pulmonary disease had a lower success rate on NPPV (without need of tracheal intubation) than COPD (35% vs 67%, p=0.01). Causes of NPPV failure were not different between COPD and patients with restrictive disease. After 12 h of NPPV, restrictive patients who succeeded with NPPV had similar respiratory rate, minute ventilation and arterial blood gas to COPD patients. At the 3rd and 12th h of NPPV, improvements in pH and $PaCO_2$ were predictive of NPPV success in COPD, but not in restrictive patients. *Conclusion:* The results of this retrospective study suggest that the effectiveness of NPPV for acute decompensation is less in patients with chronic restrictive pulmonary disease as compared to COPD.

Keywords Non-invasive pressure support ventilation · Mechanical ventilation · Chronic obstructive pulmonary disease (COPD) · Chronic restrictive pulmonary disease

Introduction

During the last decade, there has been increasing interest in non-invasive positive pressure ventilation (NPPV), particularly in intensive care and in domiciliary ventilation [1]. Randomized controlled trials have shown that NPPV decreases the need for invasive mechanical ventilation, length of hospitalization and in-hospital mortality rate in patients with chronic obstructive pulmonary disease (COPD) and acute respiratory failure (ARF) [2, 3, 4, 5]. Recent cost-effectiveness analysis suggests that adding NPPV to standard therapy may reduce the costs

of the associated care of COPD patients with severe exacerbation [6]. Indeed, COPD patients with rapid clinical deterioration should be considered for NPPV to prevent deterioration in gas exchange and the need for intubation [7]. By contrast, few data are available concerning the effectiveness of NPPV in patients with exacerbation of chronic restrictive pulmonary disease, whereas it is known that NPPV can improve quality of life and reduce the hospitalization rate for respiratory complications in stable restrictive patients [1, 8, 9, 10]. Follow-up studies from France and England have shown that nasal NPPV continuation rates were closely linked to survival in patients with chronic restrictive pulmonary disease, particularly for post-polio patients and those with kyphoscoliosis [8, 11]. Moreover, in a specific subgroup of restrictive patients (i.e. Duchenne myopathy), Simonds and colleagues showed that nasal intermittent positive pressure ventilation improves arterial blood gas and increases survival [12].

The aim of this study was to compare the efficiency of NPPV in COPD and in restrictive patients admitted to an intensive care unit (ICU) for severe exacerbation.

Patients and methods

Patients

Data for patients admitted to the medical ICU of Boucicaut Hospital with the diagnosis of exacerbation of chronic pulmonary disease were analyzed retrospectively over 3 years. Because of the observational and retrospective design of our study, no agreement by the local Institutional review board was needed accordingly to the French bioethical laws. COPD was diagnosed according to the criteria of the American Thoracic Society [13]. Chronic restrictive pulmonary disease was established on the basis of history, physical examination, radiological data (deformation of the rib cage due to tuberculosis or kyphoscoliosis on chest X-ray and unilateral phrenic paralysis on radioscopy) and respiratory function tests. Respiratory function tests were performed in a stable condition either prior to the acute episode or within 12 months following the acute episode. Exacerbation of chronic pulmonary disease was defined as exacerbation of dyspnea lasting less than 2 weeks and at least two of the following criteria: respiratory rate more than 30 breaths/min, mild encephalopathy (neurologic status score ≤3 according to Kelly and Matthay [14]), respiratory acidosis (pH <7.35), PaO_2 below 60 mmHg and $PaCO_2$ above 45 mmHg in room air.

Indication for NPPV was determined by the physician in charge and required at least two of the following clinical or physiologic criteria : signs of hypercarbia narcosis with mild asterixis, use of accessory respiratory muscles, tachypnea more than 30 breaths/min, PaO_2 below 60 mmHg on arterial blood gas analysis performed before the start of NPPV, SaO_2 less than 90%, pH less than 7.35, $PaCO_2$ above 45 mmHg or $PaCO_2$ above usual $PaCO_2$ when chronic hypercarbia was documented. Contra-indications of NPPV were: cardiac or respiratory arrest, hemodynamic instability (systolic arterial blood pressure <70 mmHg) or unstable cardiac arrhythmia, acute myocardial infarction, pulmonary thromboembolism, pneumothorax, severe encephalopathy (Kelly and Matthay score >3), facial trauma or deformity, upper airway obstruction, inability to clear respiratory secretions, high risk for aspiration. In addition, patients were excluded from analysis when they were intubated and mechanically ventilated prior to NPPV during the same ICU hospitalization stay. In cases of multiple admissions, only the first episode was considered. COPD and restrictive patients in an end-stage condition (moribund or short life expectancy) were also excluded from analysis.

The causes of ARF were defined as follow: (1) bronchitis: modification of expectoration with purulent sputum and ineffective cough; absence of systematized alveolar opacities on chest X-ray and absence of documented infection in sputum (protected specimen brush with colony forming unit $>10^3$/ml or tracheal secretions with colony forming unit $>10^7$/ml and leukocytes >25/field and epithelial cells <25/field) or blood culture. (2) Pneumonia: association of fever, leukocytosis, systematized chest crackles on physical examination, systematized alveolar opacities on chest X-ray with or without documented infection. (3) Left ventricular failure: diagnosed by physical examination and transthoracic echocardiography. (4) Adverse effects of drugs: abuse of sedative drugs, diuretics or any other drugs not recommended in chronic respiratory disease with hypoventilation. (5) Hypoventilation during systemic inflammatory response syndrome (SIRS), defined according to the American College of Chest Physicians / Society of Critical Care Medicine classification system [15], or postoperative period. (6) Primary disease: progression of the primary respiratory disease without evidence of the above-mentioned causes of ARF.

Non-invasive ventilation

In our unit, non-invasive ventilation was performed according to a standardized procedure. Patients received pressure support ventilation (PSV) via a full-face mask (CFPO KB 04 1000, Paris, France) by means of a Servo 900 C ventilator (Siemens, Sölna, Sweden). The pressure support level was adjusted to produce improved patient comfort (a decrease in respiratory rate and an increase in expiratory tidal volume) and was started with an initial inspiratory pressure of 20 cmH_2O and adjusted downward if a patient could not tolerate this level [1, 7]. FIO_2 was set to obtain a SpO_2 of 90% or more or a PaO_2 above 60 mmHg on arterial blood gas analysis when required. During PSV with Servo 900 C ventilator, the end of positive pressure and change into an inspiratory/expiratory cycle were determined by an inspiratory flow below 25% of the maximal inspiratory flow level. Positive end-expiratory pressure (PEEP) was applied and kept just above the minimal level (3–6 cmH_2O) recommended in patients with chronic restrictive pulmonary disease [1, 16] and used to counterbalance intrinsic PEEP (typically 4–6 cm H_2O) in COPD patients [7].The pressure-triggered system was set between –0.3 and –1 cmH_2O.

Non-invasive positive pressure ventilation was performed as much as possible during the first 24 h according to the course of clinical respiratory status and arterial blood gases. When clinical status and arterial blood gases improved, the duration of NPPV was progressively reduced, without reduction of the pressure support level, till complete relief of the respiratory distress was obtained. During NPPV, associate treatments were carried out by the physician in charge according to the clinical situation of each patient: antibiotics in cases of pneumonia or other documented infection, β_2-agonists and glucocorticoid when bronchospasm was present, diuretics or catecholamines for the treatment of left ventricular failure, arrest of sedative drugs or decrease of oxygen flow rate when required.

Clinical assessment (heart rate, systemic arterial blood pressure, level of consciousness, use of accessory respiratory muscles, SpO_2 and respiratory rate, tidal volume and minute ventilation assessed from the ventilator) was regularly monitored as well as patient discomfort, air leaks around the mask, gastric distension, pressure sores or facial skin necrosis. Arterial blood gases were systematically performed prior to onset (H_0) and after 12 h (H_{12}) of NPPV. Additional blood gas analysis during NPPV was carried

out between H_3 and H_6 (H_3–H_6). The success of NPPV was defined as rapid improvement in the clinical status and gas exchanges, with recovery of the earlier stable respiratory condition. Failure of NPPV was defined as the need for tracheal intubation and mechanical ventilation. Criteria for intubation were defined according to Brochard and colleagues [3] including major criteria (respiratory arrest, loss of consciousness or gasping for air, major agitation, hearth rate <50 beats/min and hemodynamic instability with systolic arterial blood pressure <70 mmHg) and minor criteria (respiratory rate >35 breaths/min and above the value of admission, arterial pH <7.30 and below the value of admission, PaO_2 below 45 mmHg despite oxygen and an increase of encephalopathy score). The presence of one major criterion or two minor criteria after the 1st h of NPPV was considered as grounds for performing intubation and mechanical ventilation.

Clinical and respiratory parameters

Age, sex, simplified acute physiologic score (SAPS II) calculated at the 24th h after ICU admission [17], weight, vital signs (systemic arterial blood pressure, heart and respiratory rates) at NPPV inclusion were recorded just before the initiation of NPPV as well as the delay between ICU admission and NPPV initiation. Arterial blood gases were collected at H_0, H_3-H_6 and H_{12}. Pressure support level, FIO_2, PEEP level, expiratory minute ventilation and respiratory rate at H_{12} were recorded on the ventilator as well as the total duration of NPPV in COPD and restrictive patients.

Statistical analysis

The results are expressed as means ± standard deviation. The statistics were calculated using StatView 4.5 software (Abacus Concepts, Berkeley, CA). Mann-Whitney's test was used to compare the COPD and restrictive groups because of the non-normal distribution of the quantitative variables and the small number of patients with chronic restrictive pulmonary disease. The χ^2 test as corrected by Yates was used to compare categorical variables. Bilateral Student's t-test for pairs was used to compare the course of respiratory parameters within each group. Bonferroni-Dunn corrections were made for statistical differences. Covariance analysis was performed to compare variation of respiratory parameters between COPD and restrictive patients. Time to occurrence of NPPV failure requiring tracheal intubation was analyzed in each group by Kaplan-Meier curves, and comparison between COPD and restrictive patients was performed by the log-rank test. A difference was considered significant when the α risk was lower than 5% ($p<0.05$).

Results

During the 3-year study period, 2,358 patients were admitted in our ICU (SAPS II=31±19, medical=93%, surgical=7%). Two hundred six patients were treated with NPPV and, among them, 84 patients fulfilled the inclusion criteria and were retained for analysis.

Characteristics of the patients

Table 1 summarizes the characteristics of the 84 patients. Sixty-four patients presented a COPD pattern whereas 20 patients had restrictive pattern. The COPD and restrictive patients did not differ in terms of age, weight, sex-ratio, use of nasal oxygen or NPPV at home, cause of ARF and initial SAPS II. The origin of COPD was smoking in 56 cases, pan lobular emphysema in 4 cases, bronchiectasis in 3 cases and cystic fibrosis in 1 case. The etiology of chronic restrictive pulmonary disease was post-tuberculosis sequelae in 10 cases, kyphoscoliosis in 4 cases, severe obesity hypoventilation syndrome (associated with central apnea) in 4 cases, unilateral

Table 1 Description of the chronic obstructive pulmonary disease (*COPD*) and restrictive (*CRPD*) patients (*SAPS II* simplified acute physiologic score, FEV_1 forced expiratory volume in 1 s, *FVC* forced vital capacity, *TLC* total lung capacity, *FRC* functional residual capacity, *NPPV* non-invasive positive pressure ventilation, *ARF* acute respiratory failure)

	COPD patients *n*=64	CRPD patients *n*=20	*p* value Mann-Whitney χ^2
Age (years)	70±13	75±9	0.07
Gender (M/F)	37/27	9/11	0.45
SAPS II	35±14	38±14	0.40
Weight (kg)	71±25	70±26	0.88
FEV_1 (% pred)	31±13	43±12	<0.01
FVC (% pred)	55±18	46±14	0.07
TLC (% pred)	91±21	57±17	<0.0001
FRC (% pred)	121±34	64±23	<0.0001
Home oxygen therapy	27/64 (42%)	9/20 (45%)	0.96
Home NPPV	7/64 (11%)	6/20 (30%)	0.08
Cause of ARF			
Bronchitis	21	10	0.26
Pneumonia	3	1	0.67
Left ventricular failure	11	1	0.16
Adverse effects of drugs	7	0	0.13
SIRS	2	1	0.56
Primary disease	20	7	0.96

Table 2 Clinical and respiratory parameters at non-invasive positive pressure ventilation (NPPV) initiation in chronic obstructive pulmonary disease (*COPD*) and chronic restrictive pulmonary disease (*CRPD*) patients

	COPD patients *n*=64	CRPD patients *n*=20	*p* value Mann-Whitney
Heart rate (beats/min)	101±19	93±16	0.08
Systolic arterial blood pressure (mmHg)	140±27	139±24	0.96
Diastolic arterial blood pressure (mmHg)	73±16	71±15	0.63
Respiratory rate (breaths/min)	27±7	29±7	0.11
Oxygen (l/min)	1.9±2.3	1.3±1.4	0.20
pH	7.27±0.09	7.28±0.09	0.78
$PaCO_2$ (mmHg)	72±15	74±17	0.66
PaO_2 (mmHg)	63±31	63±13	0.96
SaO_2	83±13	87±7	0.07
HCO_3^- (mEq/l)	32±5	33±6	0.54

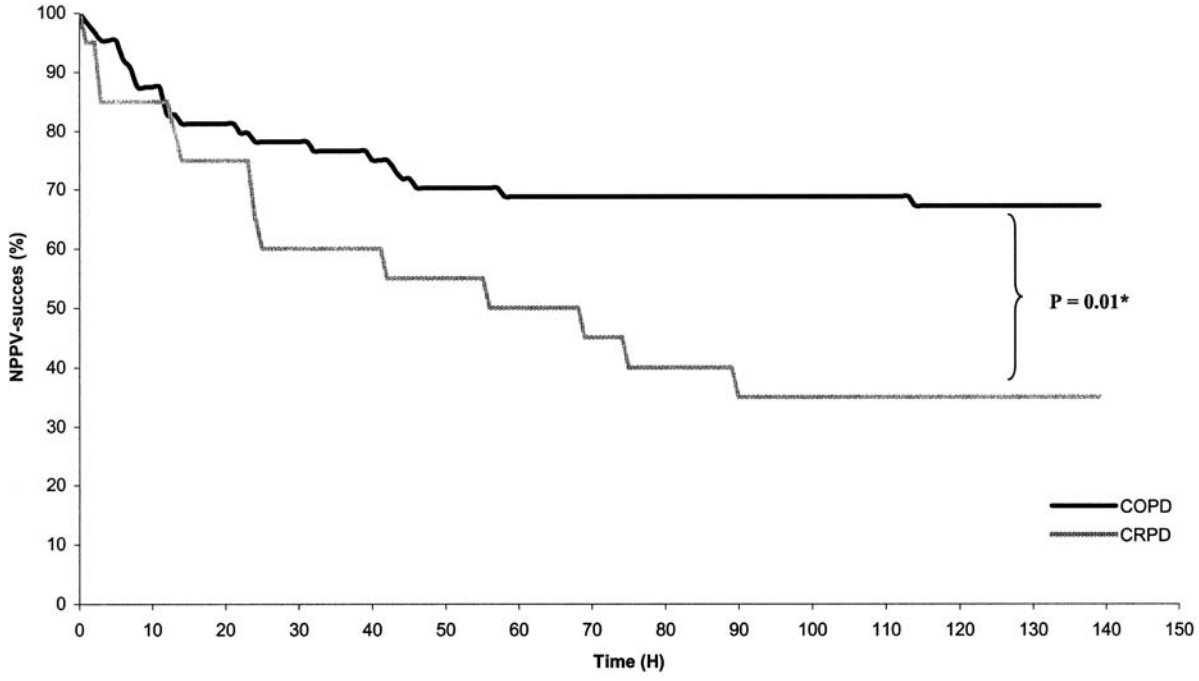

Fig. 1 Kaplan-Meier analysis of non-invasive positive pressure ventilation (*NPPV*) success in patients with chronic obstructive pulmonary disease (*COPD*; *black line*) and with chronic restrictive pulmonary disease (*CRPD; dashed line*) *By log-rank test

Table 3 Outcome and cause of non-invasive positive pressure ventilation (*NPPV*) failure in chronic obstructive pulmonary disease (*COPD*) and chronic restrictive pulmonary disease (*CRPD*) patients

	COPD patients *n*=64	CRPD patients *n*=20	*p* value Mann-Whitney χ^2
Time between ICU admission and NPPV initiation (h)	12±25	21±48	0.22
Total NPPV duration (h)	38±44	48±43	0.57
NPPV success rate	43/64 (67%)	7/20 (35%)	0.02
ICU mortality	7/64 (11%)	3/20 (15%)	0.45
Criteria for intubation:			
1 or more major criteria	19/21 (91%)	11/13 (85%)	0.49
2 or more minor criteria	2/21 (9%)	2/13 (15%)	0.67

phrenic paralysis in one case and poliomyelitis consequences in one case. At NPPV initiation, hemodynamic and respiratory parameters did not differ significantly between COPD and restrictive patients (Table 2). No major modification of chest X-ray was observed at NPPV onset.

Patient outcome

Time from ICU admission to NPPV (12±25 vs 21±48 h) and total duration of NPPV (38±44 vs 48±43 h) were not statistically different between COPD and restrictive patients. COPD patients had a higher success rate with NPPV than restrictive patients : 67 vs 35%, p=0.01 (Table 3, Fig. 1). Two restrictive patients (10%) and eight COPD patients (12.5%) failed NPPV within 12 h from the start of NPPV (Fig. 1). Patients who experienced NPPV failure were all intubated and mechanically ventilated. Differences in ICU mortality and the reasons for NPPV failure were not statistically significant between the COPD and the restrictive patients (Table 3).

Influence of cause of exacerbation, previous home non-invasive positive pressure ventilation and oxygen therapy on non-invasive positive pressure ventilation outcome

When the cause of exacerbation was the primary disease, the success rate of NPPV was higher in COPD patients than in restrictive patients (80 vs 28%, p=0.02). By con-

Table 4 Ventilatory parameters after 12 h of non-invasive positive pressure ventilation (NPPV) course in chronic obstructive pulmonary disease (*COPD*) and chronic restrictive pulmonary disease (*CRPD*) patients

	COPD Patients *n*=56/64	CRPD Patients *n*=18/20	*p* value Mann-Whitney
FIO_2 (%)	31±8	31±7	0.85
Pressure support level (cmH_2O)	18±4	19±4	0.64
PEEP (cmH_2O)	4±2	3±3	0.27
Respiratory rate (breaths/min)	22±6	21±5	0.81
Minute ventilation (l/min)	10.9±4.2	8.6±3.2	0.21

Table 5 Arterial blood gases analysis performed prior to the start of non-invasive positive pressure ventilation (*NPPV*; H_0), between the 3rd and the 6th h (H_3–H_6) of NPPV and after 12 h of NPPV (H_{12}) in patients with chronic obstructive pulmonary disease (*COPD*) and chronic restrictive pulmonary disease (*CRPD*)

Arterial blood gas analysis	COPD patients		CRPD patients	
	NPPV success	NPPV failure	NPPV success	NPPV failure
H_0	*n*=43	*n*=21	*n*=7	*n*=13
pH	7.29±0.08	7.24±0.1	7.32±0.07	7.25±0.09
$PaCO_2$ (mmHg)	70±14	77±16	68±14	77±18
PaO_2 (mmHg)	65±36	60±16	59±14	65±12
HCO_3^- (mEq/l)	32±5	31±6	33±4	33±6
H_3–H_6	*n*=43	*n*=16	*n*=7	*n*=11
pH	7.33±0.06	7.24±0.08	7.34±0.04	7.29±0.07
$PaCO_2$ (mmHg)	63±12	75±13	60±10	73±16
PaO_2 (mmHg)	77±20	73±23	72±17	69±19
HCO_3^- (mEq/l)	32±4	31±5	33±3	33±6
H_{12}	*n*=43	*n*=13	*n*=7	*n*=11
pH	7.35±0.06	7.27±0.13	7.34±0.04	7.31±0.07
$PaCO_2$ (mmHg)	62±11	74±26	61±10	69±11
PaO_2 (mmHg)	81±30	75±23	73±26	71±15
HCO_3^- (mEq/l)	33±4	32±7	33±4	33±6

trast, when the cause of exacerbation was other than progression of the primary disease, the success rate of NPPV was not significantly different between COPD and restrictive patients. Previous home NPPV or oxygen therapy did not significantly modify the success rate of NPPV in COPD and restrictive patients.

Comparison of ventilatory parameters at the twelfth hour between chronic obstructive pulmonary disease and restrictive patients

After 12 h of NPPV, respiratory rate, minute ventilation, pressure support, FIO_2 and PEEP levels were not statistically different between COPD and restrictive patients (Table 4).

Effect of non-invasive positive pressure ventilation on arterial blood gases

In COPD patients treated successfully with NPPV, pH and $PaCO_2$ improved significantly at H_3–H_6 ($p<0.001$ and $p=0.02$, respectively, Table 5) and after 12 h of NPPV ($p<0.001$) whereas PaO_2 was enhanced at H_{12} ($p<0.001$, Table 5). By contrast, only PaO_2 improved in COPD patients for whom NPPV failed after 12 h of NPPV ($p=0.01$, Table 5). Arterial blood gases differed significantly between the NPPV-success and NPPV-failure COPD patients for pH ($p<0.001$) and $PaCO_2$ ($p=0.001$) at H_3–H_6 and for pH ($p=0.01$) at H_{12}. Analysis of covariance showed that success and failure groups differed significantly in terms of variations of pH (ΔpH = 0.06±0.08 vs 0.03±0.11, $p=0.01$) and $PaCO_2$ ($\Delta PaCO_2$ = −8.5±13.9 vs −0.6±21.6 mmHg, $p=0.02$) between H_0 and H_{12}.

In restrictive patients successfully treated with NPPV, arterial blood gases were not significantly improved at H_3–H_6 and H_{12} (Table 5). In restrictive patients with unsuccessful NPPV, pH was significantly improved ($p=0.04$) but not $PaCO_2$, PaO_2 and $HCO3^-$ after 12 h of NPPV (Table 5). Arterial blood gases performed at H_0, H_3-H_6 and H_{12} did not differ significantly between the NPPV-success and NPPV-failure restrictive groups (Table 5). Analysis of covariance showed no significant difference between variations of pH, $PaCO_2$, PaO_2, HCO_3^- in NPPV-success and in NPPV-failure restrictive groups.

Discussion

In this study, the success rate of NPPV in chronic restrictive pulmonary disease patients with severe exacerbation was significantly lower than in decompensated COPD patients. Moreover, in contrast with COPD patients, the course of arterial blood gas parameters was not predictive of NPPV success or failure in restrictive patients. Our results suggest that the decrease of chest wall compliance related to deformation of the rib cage in chronic restrictive pulmonary disease could influence the respiratory behavior of these patients during pressure support ventilation (PSV) with facial mask and could impair the NPPV success rate.

In the past decade, large randomized studies have underlined the efficiency of NPPV in COPD patients with acute respiratory failure (ARF) [2, 3, 4, 5]. These studies reported a success rate of NPPV from 74 to 100%. Other large retrospective studies showed a success rate of NPPV (full-face mask, PSV ± PEEP) from 66 to 92% and a NPPV duration from 25 to 72 h [18, 19, 20, 21, 22]. Our results are in agreement with these works since we found a NPPV success rate of 67% and a NPPV mean duration of 38 h in our 64 COPD patients. However, few studies focused on the effectiveness of NPPV in chronic restrictive pulmonary disease patients with acute respiratory failure. Elliott and colleagues reported two successes of NPPV (nasal mask, volume-assisted controlled ventilation) in three cases of restrictive patient with severe exacerbation [23]. Meduri and colleagues reported two decompensated patients with chronic restrictive pulmonary disease treated by NPPV (facial mask, PSV) and one success [19].

The same authors collected three other cases of restrictive patients with ARF and reported success of NPPV (facial mask, PSV) in one case [22]. Benhamou and colleagues found a NPPV success rate of 50% in two restrictive patients (nasal mask, volume-assisted controlled ventilation) [24]. In a randomized trial, Wysocki and colleagues counted only two restrictive patients amongst their non-COPD patients with ARF [25]. These two patients were treated by NPPV (facial mask, PSV), but one needed intubation. To our knowledge, our study is the first to gather a relatively large sample of chronic restrictive pulmonary disease patients treated with NPPV for ARF, thus allowing the assessment of the usefulness of NPPV in this clinical situation. Our success rate for NPPV in this population (35%) was close to the data reported in the above-mentioned papers.

In comparison to the COPD patients, the efficiency of NPPV in restrictive patients appears to be poor. Demographic data, home oxygen therapy, initial severity or explicit cause of exacerbation did not explain the difference in outcome. However, in our study, COPD patients had a higher rate of left ventricular failure diagnosed by echocardiography than the restrictive patients, even if the difference did not reach statistical significance because of the small cohort. Indeed, NPPV has been shown to improve oxygenation and hypercapnia, and reduce the need of tracheal intubation in patients with acute congestive heart failure, except myocardial infarction, emphasizing a possible bias in our retrospective analysis [26, 27, 28]. In addition, we found that COPD patients had a shorter delay from ICU admission to NPPV and lower rate of previous home NPPV than restrictive patients, but these differences did not reach the statistical threshold (Tables 1 and 3) and the plasma bicarbonate concentrations (a reflection of the degree of chronic hypercapnia [18]) on admission were similar in the two groups. Chronic restrictive pulmonary disease patients with home NPPV may have fewer difficulties in undergoing NPPV in acute settings, but we found no difference between the success rate of NPPV in restrictive patients with NPPV at home and those without previous NPPV.

Interestingly, the difference in NPPV outcome between restrictive and COPD patients was statistically significant when acute respiratory failure was only related to the underlying respiratory disease. This could suggest that the respiratory behavior of restrictive patients towards NPPV differs from that of COPD patients. In COPD patients, improvement in arterial blood gases during NPPV is due to higher alveolar ventilation corresponding to a ventilatory pattern with increase in minute ventilation and decrease in respiratory breath [29, 30]. Our chronic restrictive pulmonary disease patients most likely showed a decrease in their chest wall compliance due to abnormality of their rib cages as a result of post-tuberculosis sequelae, kyphoscoliosis or severe hypoventilation syndrome. During PSV, a decrease of respiratory compliance involves a reduction in tidal volume due to a decrease of inspiratory flow [31]. However, we observed no significant difference in minute ventilation and respiratory rate between COPD and restrictive patients after 12 h of NPPV (Table 4).

When PSV is used, a rapid decline of inspiratory flow involves the untimely end of the inspiratory cycle [1, 31]. In chronic restrictive pulmonary disease patients with impaired chest wall compliance, PSV could markedly reduce the inspiratory time, which is a major determinant of the respiratory muscle weakness [32]. Accordingly, this hypothesis could explain the difference in NPPV success rate between restrictive patients and COPD patients who have normal or increased respiratory compliance. But no precise measurements of chest wall compliance were performed in our patients with chronic restrictive pulmonary disease. Further investigations are needed to confirm this assumption.

In COPD patients, Meduri and colleagues showed that correction of $PaCO_2$ (−16% vs initial value) and pH (>7.30) after 2 h of NPPV was a good indicator of NPPV success [19]. We confirm, in part, these results in our COPD patients. Indeed, after 3 h of NPPV, improvement

in pH and $PaCO_2$ was achieved in the COPD success group, in contrast to the COPD failure group (Table 5). Correction of PaO_2 does not appear predictive of the success or failure of NPPV (Table 5). By contrast, variations of arterial blood gas parameters during NPPV in restrictive patients were different from COPD patients. We found that variations of pH, $PaCO_2$ and PaO_2 were similar between the success and failure groups in patients with chronic restrictive pulmonary disease (Table 5). A distinct respiratory behavior pattern towards PSV could also explain these different results. In addition, COPD and restrictive patients who failed NPPV had a trend towards lower pH than NPPV success patients (Table 5). This may be due in part to higher $PaCO_2$ in the NPPV failure group, but lower pH may be a reflection of greater tissue hypoperfusion and severity in this group. Unfortunately, we did not measure blood arterial lactate in our study.

Our study have several limitations. The major limitation is the retrospective design because co-interventions were not standardized. The retrospective design of this study does not exclude the possibility of having pre-selected patients according to subjective decisions or according to different inclusion criteria of the physicians in charge. The relatively small CRPD population leads to a lack of power analysis and caution is needed in extrapolating our results beyond our patient recruitment and standard procedures of care.

In summary, the results of this retrospective study suggest that the effectiveness of NPPV for acute decompensation is reduced in patients with chronic restrictive pulmonary disease as compared to COPD. Reduction of chest wall compliance could limit the resolution of respiratory muscle weakness by PSV and may explain this difference, at least in part. Further investigations are needed to confirm the results of our analysis.

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